Set	Items	Description
S1	461	CYR61
S2	38	CYR(W)61
S3	17	CYSTEINE(W)RICH(W)PROTEIN(W)61
S4	494	S1 OR S2 OR S3
S5	117041	INTEGRIN? ?
S6	640243	FIBROBLAST? ?
S7	1722963	GEL OR GELATIN OR GELATINIZE? ?
S8	11825	S5 AND S6
S9	40	S4 AND S8
S10	31	S9 NOT PY>2000
S11	18	RD (unique items)
S12	5	S11 AND S7
S13	13	S11 NOT S12
S14	74	S4 NOT PY>1996
S15	24	RD (unique items)
S16	38684	HEPARAN(W)(SULFATE OR SULPHATE)
S17	10894	S6 AND S16
S18	695712	MIGRATE? ? OR MIGRATION
S19	1240	S17 AND S18
S20	275	S5 AND S19
S21	80	S20 NOT PY>1996
S22	79	RD (unique items)
S23	33	S7 AND S22
S24	593701	MATRIGEL OR COLLAGEN OR FIBRIN
S25	46	S22 NOT S23
S26	660	S6 AND S16 AND S18 AND S24
S27	278	S26 NOT PY>1996
S28	253	RD (unique items)
S29	47	S28 AND S5
S30	220	AU="LAU L" OR AU="LAU L F"
S31	19	AU="LAU L.-F."
S32	46	AU="LAU LESTER F"
S33	285	S30 OR S31 OR S32
S34	32	S4 AND S33
S35	10	S34 NOT PY>1996
S36	7	RD (unique items)

?

04160189

MATRIGEL

INTL CLASS: 1 (Chemicals)
U.S. CLASS: 6 (Chemicals & Chemical Compositions)
STATUS: Registered; Section 8 & 15 - Accepted & Acknowledged
GOODS/SERVICES: BIOLOGICAL CELL CULTURE SUBSTRATE
SERIAL NO.: 74-160,189
REG. NO.: 1,721,244
REGISTERED: October 6, 1992
FIRST USE: September 1986 (Intl Class 1)
FIRST COMMERCE: September 1986 (Intl Class 1)
FILED: April 24, 1991
PUBLISHED: July 14, 1992
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ORIGINAL APPLICANT: COLLABORATIVE RESEARCH, INC. (Massachusetts Corporation), TWO OAK PARK, BEDFORD, MA (Massachusetts), 01730, USA (United States of America)
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Reel/Frame: 0814/0796
Recorded: September 20, 1991
Brief: ASSIGNS THE ENTIRE INTEREST AND GOOD WILL
FILING CORRESPONDENT: ROBERT M. HALLENBECK, DAVINA BOWMAN, 1 BECTON DR, FRANKLIN LAKES NJ 07417

?

12/3,AB/1 (Item 1 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00743044
HUMAN CANCER ASSOCIATED GENE SEQUENCES AND POLYPEPTIDES
SEQUENCES ET POLYPEPTIDES GENIQUES ASSOCIES AU CANCER CHEZ L'HOMME
Patent Applicant/Assignee:
HUMAN GENOME SCIENCES INC, 9410 Key West Avenue, Rockville, MD 20850, US, US (Residence), US (Nationality), (For all designated states except: US)
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Legal Representative:
WALES Michele M, Human Genome Sciences, Inc., 9410 Key West Avenue, Rockville, MD 20850, US
Patent and Priority Information (Country, Number, Date):
Patent: WO 200055350 A1 20000921 (WO 0055350)
Application: WO 2000US5882 20000308 (PCT/WO US0005882)
Priority Application: US 99124270 19990312
Designated States: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW
(EP) AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
(OA) BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG
(AP) GH GM KE LS MW SD SL SZ TZ UG ZW
(EA) AM AZ BY KG KZ MD RU TJ TM
Publication Language: English
Filing Language: English
Fulltext Word Count: 223528

English Abstract
This invention relates to newly identified tissue specific cancer associated polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "cancer antigens", and to the complete gene sequences associated therewith and to the expression products thereof, as well as the use of such tissue specific cancer antigens for detection, prevention and treatment of tissue specific disorders, particularly the presence of cancer. This invention relates to the cancer antigens as well as vectors, host cells, antibodies directed to cancer antigens and recombinant and synthetic methods for producing the same. Also provided are diagnostic methods for diagnosing and treating, preventing and/or prognosing tissue specific disorders, including cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of cancer antigens of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and/or function of the polypeptides of the present invention.

French Abstract
Cette invention porte sur des polynucleotides recemment identifies et associes au cancer specifique d'un tissu, et sur les polypeptides codes par ces polynucleotides et connus collectivement sous le nom <=d'antigenes du cancer>=. L'invention porte egalement surles sequences geniques completes associees et sur leurs produits d'expression, ainsi

que sur l'utilisation de ces antigenes du cancer specifique d'un tissu dans la detection, la prevention et le traitement des pathologies specifiques d'un tissu telles que le cancer. Cette invention porte sur les antigenes du cancer, ainsi que sur les vecteurs, les cellules hotes, les anticorps diriges contre les antigenes du cancer et sur des procedes recombinants et synthetiques de production de ces anticorps. L'invention porte egalement sur des procedes de diagnostic permettant de diagnostiquer et traiter, prevenir et/ou etablir un pronostic de pathologies specifiques d'un tissu telles que le cancer, et sur des procedes therapeutiques visant a traiter ces pathologies. Cette invention porte en outre sur des procedes de recherche automatique visant a identifier des agonistes et des antagonistes des antigenes du cancer, et sur des procedes et/ou des compositions visant a inhiber la production et/ou la fonction des polypeptides de cette invention.

12/3,AB/2 (Item 2 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00683145
POLYNUCLEOTIDE POPULATION ISOLATED FROM NON-METASTATIC AND METASTATIC BREAST TUMOR TISSUES
POPULATION DE POLYNUCLEOTIDES ISOLES DE TISSUS MAMMAIRES TUMORAUX METASTATIQUES OU NON METASTATIQUES
Patent Applicant/Assignee:
GENZYME CORPORATION, GENZYME CORPORATION , Metrowest Place, 15 Pleasant Street Connector, Framingham, MA 01701-9322 , US
ROBERTS Bruce L
SHANKARA Srinivas
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ROBERTS Bruce L
SHANKARA Srinivas
Patent and Priority Information (Country, Number, Date):
Patent: WO 9965928 A2 19991223
Application: WO 99US13647 19990618 (PCT/WO US9913647)
Priority Application: US 9890039 19980619; US 9890040 19980619; US 9890041 19980619; US 9889853 19980619; US 9889997 19980619
Designated States: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW GH GM KE LS MW SD SL SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG
Publication Language: English
Filing Language: English
Fulltext Word Count: 42035

English Abstract
NotAvailable

12/3,AB/3 (Item 3 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00657448
ANGIOGENESIS TARGETING MOLECULES
MOLECULES CIBLANT L'ANGIOGENESE

Patent Applicant/Assignee:
RESOLUTION PHARMACEUTICALS INC, RESOLUTION PHARMACEUTICALS INC. , 6850 Goreway Drive, Mississauga, Ontario LV4 1V7 , CA
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Patent and Priority Information (Country, Number, Date):
Patent: WO 9940947 A2 19990819
Application: WO 99CA101 19990211 (PCT/WO CA9900101)
Priority Application: US 9874420 19980211
Designated States: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW GH GM KE LS MW SD SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG
Publication Language: English
Filing Language: English
Fulltext Word Count: 16792

English Abstract
The present invention relates to compounds that are effective for targeting sites of angiogenesis for diagnostic and therapeutic purposes. The compounds are of the Formula (I): A-(B)n-C, wherein A is a chelator moiety capable of complexing a radionuclide metal or a moiety capable of binding to a halogen; B is a spacer group; C is an angiogenesis targeting molecule; and n is selected from the integers 0 and 1. The invention also relates to a method of imaging sites of angiogenesis and treating patients through the administration of the compounds of the present invention.

French Abstract
La presente invention concerne des composes efficaces pour cibler des sites d'angiogenese C des fins diagnostiques et therapeutiques. Les composes ont la formule suivante (I): A-(B)n-C, dans laquelle A represente une fraction de chelateur capable de complexer un metal de radionucleide ou une fraction capable de se fixer C un halogene; B represente un groupe espaceur; C represente une molecule ciblant l'angiogenese et n est choisi entre les nombres entiers 0 et 1. L'invention concerne egalement un procede d'imagerie de sites d'angiogenese et de traitement de patients par l'administration des composes de la presente invention.

12/3,AB/4 (Item 4 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00533203
EXTRACELLULAR MATRIX SIGNALLING MOLECULES
MOLECULES DE SIGNALISATION DE MATRICE EXTRACELLULAIRE
Patent Applicant/Assignee:
MUNIN CORPORATION
LAU Lester F
Inventor(s):

LAU Lester F
Patent and Priority Information (Country, Number, Date):
Patent: WO 9733995 A2 19970918
Application: WO 97US4193 19970314 (PCT/WO US9704193)
Priority Application: US 9613958 19960315
Designated States: AL AM AT AU AZ BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU IL IS JP KP KR KZ LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SK TJ TM TR TT UA UG US UZ VN GH KE LS MW SD SZ UG AM AZ BY KG KZ MD RU AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA MR NE SN TD TG
Publication Language: English
Fulltext Word Count: 39033

English Abstract
Polynucleotides encoding mammalian ECM signalling molecules affecting the cell adhesion, migration, and proliferation activities characterizing such complex biological processes as angiogenesis, chondrogenesis, and oncogenesis, are provided. The polynucleotide compositions include DNAs and RNAs comprising part, or all, of an ECM signalling molecule coding sequence, or biological equivalents. Polypeptide compositions are also provided. The polypeptide compositions comprise mammalian ECM signalling molecules, peptide fragments, inhibitory peptides capable of interacting with receptors for ECM signalling molecules, and antibody products recognizing **Cyr61** . Also provided are methods for producing mammalian ECM signalling molecules. Further provided are methods for using mammalian ECM signalling molecules to screen for, and/or modulate, disorders associated with angiogenesis, chondrogenesis, and oncogenesis; ex vivo methods for using mammalian ECM signalling molecules to prepare blood products are also provided.

Japanese Abstract
L'invention porte sur des polynucleotides codant des molecules de signalisation ECM de mammiferes influant sur les activites d'adhesion, de migration, et de proliferation caracteristiques de processus biologiques complexes tels que l'angiogenese, la condrogenese, et l'oncogenese. Les compositions de polynucleotides comportent des ADN et des ARN comprenant en partie ou en totalite une sequence codant une molecule de signalisation ECM ou des equivalents biologiques. L'invention porte egalement sur des compositions de polypeptides. Lesdites compositions de polypeptides comprenant des molecules de signalisation de matrices extracellulaires de mammiferes, des fragments de peptides, des peptides inhibiteurs capables d'interagir avec les recepteurs des molecules de signalisation de matrices extracellulaires, et des anticorps reconnaissant la proteine **Cyr61** . L'invention porte en outre sur des procedes de production de molecules de signalisation de matrices extracellulaires; sur des procedes d'utilisation desdites molecules pour depister et/ou traiter differents troubles lies a l'angiogenese, la condrogenese, et l'oncogenese, et sur des procedes ex vivo d'utilisation desdites molecules pour la preparation de produits sanguins.

12/3,AB/5 (Item 1 from file: 654)
DIALOG(R)File 654:US PAT.FULL.

02874538
Utility
INDUCTION OF TISSUE, BONE OR CARTILAGE FORMATION USING CONNECTIVE TISSUE GROWTH FACTOR
[Administering as wound healing agent; treating osteoporosis, osteoarthritis, and osteochondrytis]

PATENT NO.: 5,837,258
ISSUED: November 17, 1998 (19981117)
INVENTOR(s): Grotendorst, Gary R., Miami, FL (Florida), US (United States of America)
ASSIGNEE(s): University of Miami, (A U.S. Company or Corporation), Miami, FL (Florida), US (United States of America)
University of South Florida, (A U.S. Company or Corporation), Tampa, FL (Florida), US (United States of America)
[Assignee Code(s): 16948; 55026]
APPL. NO.: 8-656,393
FILED: May 31, 1996 (19960531)

STATEMENT OF RELATED CASES

This application is related to and is a continuation-in-part application of Serial Number 08-459,717, entitled "Connective Tissue Growth Factor," filed Jun. 2, 1995, which is a continuation-in-part application of Ser. No. 08-386,680, filed on Feb. 10, 1995, having the same title, now issued as U.S. Pat. No. 5,585,270, which is a divisional application of Ser. No. 08-167,628, filed Dec. 14, 1993, now issued as U.S. Pat. No. 5,408,040, which is a continuation of Ser. No. 07-752,427, filed on Aug. 30, 1991, now abandoned.

The information disclosed in this Specification was made in part with Government support by grant no. GM 37223, awarded by the National Institute of Health. The government may have certain rights in the invention disclosed in this Specification.

FULL TEXT: 1383 lines

ABSTRACT

The present invention relates to novel methods and compositions related to the administration of connective tissue growth factor, alone or in combination with other growth factors, compositions or compounds, to induce the formation of connective tissue, including bone, cartilage, and the skin.
?

15/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08920011 96239486 PMID: 8657105
Cyr61 , **a product of a growth factor-inducible immediate-early gene, promotes cell proliferation, migration, and adhesion.**
Kireeva ML; MO FE; Yang GP; Lau LF
Department of Genetics, University of Illinois College of Medicine, Chicago, 60607-7170, USA.
Molecular and cellular biology (UNITED STATES) Apr 1996, 16 (4) p1326-34, ISSN 0270-7306 Journal Code: NGY
Contract/Grant No.: R01 CA46565-08, CA, NCI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
cyr61 was first identified as a growth factor-inducible immediate-early gene in mouse fibroblasts. The encoded **Cyr61** protein is a secreted, cystein-rich heparin-binding protein that associates with the cell surface and the extracellular matrix, and in these aspects it resembles the Wnt-1 protein and a number of known growth factors. During embryogenesis, **cyr61** is expressed most notably in mesenchymal cells that are differentiating into chondrocytes and in the vessel walls of the developing circulatory system. **cyr61** is a member of an emerging gene family that encodes growth regulators, including the connective tissue growth factor and an avian proto-oncoprotein, Nov **cyr61** also shares sequence similarities with two Drosophila genes, twisted gastrulation and short gastrulation, which interact with decapentaplegic to regulate dorsal-ventral patterning. In this report we describe the purification of the **Cyr61** protein in a biologically active form, and we show that purified **Cyr61** has the following activities: (i) it promotes the attachment and spreading of endothelial cells in a manner similar to that of fibronectin; (ii) it enhances the effects of basic fibroblast growth factor and platelet-derived growth factor on the rate of DNA synthesis of fibroblasts and vascular endothelial cells, although it has no detectable mitogenic activity by itself; and (iii) it acts as a chemotactic factor for fibroblasts. Taken together, these activities indicate that **Cyr61** is likely to function as an extracellular matrix signaling molecule rather than as a classical growth factor and may regulate processes of cell proliferation, migration, adhesion, and differentiation during development.

15/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08564942 95348101 PMID: 7622488
Glucocorticoid-attenuated response genes encode intercellular mediators, including a new C-X-C chemokine.
Smith JB; Herschman HR
Division of Neonatology, UCLA School of Medicine 90095, USA.
Journal of biological chemistry (UNITED STATES) Jul 14 1995, 270 (28) p16756-65, ISSN 0021-9258 Journal Code: HIV
Contract/Grant No.: GM24797, GM, NIGMS
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
A major part of the anti-inflammatory effect of glucocorticoids is attributable to their attenuation of the induction of genes whose products mediate intercellular interactions, e.g. cytokines and the inducible forms of prostaglandin synthase and nitric oxide synthase. We hypothesized that (i) there exists a class of immediate-early/primary response genes whose induction by inflammatory agents, mitogens, and other stimuli is attenuated

by glucocorticoids, and (ii) the products of these glucocorticoid-attenuated response genes (GARGs) function predominantly in paracrine cell processes. We constructed a lambda cDNA library from transforming growth factor beta 1-pretreated murine Swiss 3T3 cells stimulated with lipopolysaccharide (LPS) or serum in the presence of cycloheximide, screened 15,000 plaques by differential hybridization, and cloned 12 LPS-induced, dexamethasone-attenuated cDNAs. Seven were previously known. Six of these encode intercellular mediators (thrombospondin-1, MCSF, JE/MCP-1, MARC/fic/MCP-3, crg2/IP-10, and **cyr61**); one encodes a protein of unknown function (IRG2). Thus, a large majority of these GARG cDNAs encode intercellular mediators, as hypothesized. Of the five GARG cDNAs not previously known, one encodes a novel member of the CXC chemokine family, designated LIX (LPS-induced CXC chemokine). The predicted LIX protein has a 40-amino acid signal sequence and a 92-amino acid mature peptide with a distinctive COOH-terminal region. Surprisingly, segments of the 3'-untranslated regions of LIX and two other CXC chemokines have substantially greater nucleotide sequence homology than do their coding regions. These segments may perform an unknown regulatory function. The LIX message is strongly induced by LPS in fibroblasts, but not in macrophages, suggesting that LIX may participate in the recruitment of inflammatory cells by injured or infected tissue.

15/3,AB/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08075927 93327926 PMID: 7687569
The modular architecture of a new family of growth regulators related to connective tissue growth factor.
Bork P
Max-Delbruck-Centre for Molecular Medicine, Berlin-Buch, Germany.
FEBS letters (NETHERLANDS) Jul 26 1993, 327 (2) p125-30, ISSN 0014-5793 Journal Code: EUH
Languages: ENGLISH
Document type: Journal Article; Review; Review, Tutorial
Record type: Completed
Recently, several groups have characterized and sequenced members of a new family of growth regulators (originally called cef10, connective tissue growth factor, fisp-12, **cyr61** , or, alternatively, beta IG-M1 and beta IG-M2), all of which belong to immediate-early genes expressed after induction by growth factors or certain oncogenes. Sequence analysis of this family revealed the presence of four distinct modules. Each module has homologues in other extracellular mosaic proteins such as Von Willebrand factor, slit, thrombospondins, fibrillar collagens, IGF-binding proteins and mucins. Classification and analysis of these modules suggests the location of binding regions and, by analogy to better characterized modules in other proteins, sheds some light onto the structure of this new family.

15/3,AB/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07741755 91229699 PMID: 2029337
Identification of a gene family regulated by transforming growth factor-beta.
Brunner A; Chinn J; Neubauer M; Purchio AF
Bristol-Myers Squibb Pharmaceutical Research Institute, Seattle, WA 98121.
DNA and cell biology (UNITED STATES) May 1991, 10 (4) p293-300, ISSN 1044-5498 Journal Code: AF9
Languages: ENGLISH

Document type: Journal Article
Record type: Completed

We have identified two related genes whose mRNAs are increased after treatment with transforming growth factor-beta (TGF-beta 1). Mouse AKR-2B cells were treated with TGF-beta 1 in the presence of cyclohexamide and a cDNA library was subjected to differential screening. Several TGF-beta-induced genes (beta IG) were isolated and two of these, beta IG-M1 and beta IG-M2, were characterized. beta IG-M1 and beta IG-M2 RNAs were significantly increased after TGF-beta 1 treatment and both were superinduced in the presence of cyclohexamide. cDNA sequence analysis of beta IG-M1 showed that it encoded a 379-amino-acid protein which was 81% homologous to CEF-10, a v-src and TPA-inducible gene, and identical to **cyr61** , a gene induced by serum in growth-arrested BALB-3T3 cells. cDNA sequence analysis of beta IG-M2 showed that it encoded a 348-amino-acid protein that was 50% homologous to beta IG-M1. Thirty-eight cysteine residues are conserved between beta IG-M1 and beta IG-M2, which are clustered at the amino and carboxy ends: The middle regions of the two proteins are cysteine free and display the highest degree of nonhomology. Both proteins contain an amino-terminal cysteine-rich motif common to insulin-like growth factor binding proteins and a carboxy-terminal domain with strong homology to a motif found near the carboxy-terminal of the malarial circumsporozoite protein which may be involved in cell adhesion. The regulation of mRNA encoding these proteins by TGF-beta 1 suggests that they may be involved in mediating some of the pleiotropic effects of this multipotent modulator of cell growth and differentiation.

15/3,AB/7 (Item 7 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07661183 93041389 PMID: 1419914
Expression of the growth factor-inducible immediate early gene cyr61 **correlates with chondrogenesis during mouse embryonic development.**
O'Brien TP; Lau LF
Department of Genetics, University of Illinois College of Medicine, Chicago 60612.
Cell growth & differentiation (UNITED STATES) Sep 1992, 3 (9) p645-54, ISSN 1044-9523 Journal Code: AYH
Contract/Grant No.: CA46565, CA, NCI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed

cyr61 is a growth factor-inducible immediate early gene initially identified in serum-stimulated mouse fibroblasts. It encodes a member of an emerging family of cysteine-rich secreted proteins that includes a connective tissue growth factor. We show here that **cyr61** is expressed in the developing mouse embryo and extraembryonic tissues. In the placenta, **cyr61** is expressed in regions of trophoblastic origin, including the ectoplacental cone and the trophoblastic giant cells. In the midgestation embryo, **cyr61** is expressed in the smooth muscle vessel walls of the arterial circulatory system. Most notably, expression is found in developing cartilaginous elements, including the limbs, ribs, and prevertebrae. In addition, regions of the chondrocranium and craniofacial elements, such as Meckel's cartilage, also express **cyr61** . Thus, **cyr61** transcript is found in mesenchymal cells of both mesodermal and ectodermal origin during their differentiation into chondrocytes. The temporal and spatial regulation of **cyr61** expression and the biochemical features of its encoded protein suggest that **cyr61** may be important for the normal growth, differentiation, or morphogenesis of the cartilaginous skeleton of the embryo.

15/3,AB/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06812963 92144441 PMID: 1782153
Cyr61 , **product of a growth factor-inducible immediate early gene, is associated with the extracellular matrix and the cell surface.**
Yang GP; Lau LF
Department of Genetics, University of Illinois College of Medicine, Chicago 60612.
Cell growth & differentiation (UNITED STATES) Jul 1991, 2 (7) p351-7 , ISSN 1044-9523 Journal Code: AYH
Contract/Grant No.: RO1 CA46565, CA, NCI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
cyr61 is a specific target for activation by platelet-derived growth factor and fibroblast growth factor and is inducible by the oncogene v-src. It is a member of the class of immediate early genes that includes those encoding protooncogene products, transcription factors, and cytokines. We have previously characterized the synthesis and degradation of the **cyr61** -encoded mRNA and protein. Although the deduced **Cyr61** protein sequence contains an NH2-terminal secretory signal, it is not detectable in the conditioned medium of serum-stimulated cells. We show here that in rapidly growing cell cultures, newly synthesized **Cyr61** is secreted and is associated with both the extracellular matrix and the cell surface. In contrast, **Cyr61** secreted in serum-stimulated quiescent cells is directed to the cell surface and is hot incorporated into the extracellular matrix. Once associated with the extracellular matrix, **Cyr61** has a half-life of greater than 24 h, whereas intracellular and cell surface-associated **Cyr61** has an apparent half-life of approximately 30 min. Furthermore, **Cyr61** appears to bind heparin with high affinity. These observations suggest similarities among **Cyr61** , the fibroblast growth factors (heparin-binding growth factors), and the protooncogene product Int-1 and are consistent with the hypothesis that **Cyr61** plays a role in cell-cell communication involving the interaction of neighboring cells.

15/3,AB/10 (Item 10 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06769746 91288203 PMID: 2062642
Promoter function and structure of the growth factor-inducible immediate early gene cyr61.
Latinkic BV; O'Brien TP; Lau LF
Department of Genetics, University of Illinois College of Medicine, Chicago 60612.
Nucleic acids research (ENGLAND) Jun 25 1991, 19 (12) p3261-7, ISSN 0305-1048 Journal Code: O8L
Contract/Grant No.: R01 CA52220, CA, NCI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
cyr61 is an immediate early gene that is transcriptionally activated in 3T3 fibroblasts by serum, platelet-derived growth factor, fibroblast growth factor, and the tumor promoter TPA with kinetics similar to the induction of c-fos. **cyr61** encodes a secreted protein that is associated with the cell surface and the extracellular matrix, and may play a role in cell-cell communication. We report here the complete nucleotide sequence of the mouse **cyr61** gene, which contains four short introns. The transcription start site was mapped by S1 nuclease and primer extension analyses. A 2 kb 5'

flanking DNA fragment functions as a serum-inducible promoter. This DNA fragment contains a poly(CA) sequence that can adopt the Z DNA form. In addition, it contains a sequence that resembles the serum response element (SRE) originally identified in the c-fos promoter. We show that deletion of the cry61 SRE-like sequence abrogates serum inducibility. Furthermore, this SRE-like sequence is sufficient to confer serum and growth factor inducibility when linked to a basal promoter, and binds the 67 kD serum response factor in vitro. We conclude that the **cyr61** SRE functions as a serum response element and may account for the coordinate activation of **cyr61** and c-fos.

15/3,AB/11 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

06643886 90287146 PMID: 2355916
Expression of cyr61, **a growth factor-inducible immediate-early gene.**
O'Brien TP; Yang GP; Sanders L; Lau LF
Department of Genetics, University of Illinois College of Medicine, Chicago 60612.
Molecular and cellular biology (UNITED STATES) Jul 1990, 10 (7) p3569-77, ISSN 0270-7306 Journal Code: NGY
Contract/Grant No.: R01 CA46565, CA, NCI; R01 CA52220, CA, NCI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
A set of immediate-early genes that are rapidly activated by serum or purified platelet-derived growth factor in mouse 3T3 fibroblasts has been previously identified. Among these genes, several are related to known or putative transcription factors and growth factors, supporting the notion that some of these genes encode regulatory molecules important to cell growth. We show here that a member of this set of genes, **cyr61** (originally identified by its cDNA 3CH61), encodes a 379-amino-acid polypeptide rich in cysteine residues. **cyr61** can be induced through protein kinase C-dependent and -independent pathways. Unlike many immediate-early genes that are transiently expressed, the **cyr61** mRNA is accumulated from the G0/G1 transition through mid-G1. This expression pattern is due to persistent transcription, while the mRNA is rapidly turned over during the G0/G1 transition and in mid-G1 at the same rate. In logarithmically growing cells, the **cyr61** mRNA level is constant throughout the cell cycle. **Cyr61** contains an N-terminal secretory signal sequence; however, it is not detected in the culture medium by immunoprecipitation. **Cyr61** is synthesized maximally at 1 to 2 h after serum stimulation and has a short half-life within the cell.

15/3,AB/12 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10774773 BIOSIS NO.: 199799395918
Cloning of human homolog of cyr61 **and characterization of its biological activities.**
AUTHOR: Kolesnikova T V; Lau L F
AUTHOR ADDRESS: Dep. Genet., Univ. Illinois, Chicago, IL 60607**USA
JOURNAL: Molecular Biology of the Cell 7 (SUPPL.):p415A 1996
CONFERENCE/MEETING: Annual Meeting of the 6th International Congress on Cell Biology and the 36th American Society for Cell Biology San Francisco, California, USA December 7-11, 1996
ISSN: 1059-1524
RECORD TYPE: Citation

LANGUAGE: English
1996

15/3,AB/13 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

08280938 BIOSIS NO.: 000043047011
THE IMMEDIATE EARLY GENE CYR61 **ENCODES A HEPARIN BINDING PROTEIN WHOSE IN-VIVO EXPRESSION CORRELATES WITH CHONDROGENESIS**
AUTHOR: YANG G P; O'BRIEN T P; ABLER A S; LAU L F
AUTHOR ADDRESS: DEP. GENET., UNIV. ILL. COLL. MED., CHICAGO, ILL. 60612, USA.
JOURNAL: KEYSTONE SYMPOSIUM ON GROWTH AND DIFFERENTIATION FACTORS IN VERTEBRATE DEVELOPMENT, KEYSTONE, COLORADO, USA, APRIL 3-10, 1992. J CELL BIOCHEM SUPPL 0 (16 PART F). 1992. 104. 1992
CODEN: JCBSD
DOCUMENT TYPE: Meeting
RECORD TYPE: Citation
LANGUAGE: ENGLISH
1992

15/3,AB/15 (Item 1 from file: 266)
DIALOG(R)File 266:FEDRIP

00311552
IDENTIFYING NO.: 5R01GM37223-14 AGENCY CODE: CRISP
CELLULAR AND MOLECULAR PATHOBIOLOGY OF WOUND REPAIR
PRINCIPAL INVESTIGATOR: GROTENDORST, GARY R
ADDRESS: UNIVERSITY OF MIAMI 1600 N W 10TH AVENUE MIAMI, FL 33136
PERFORMING ORG.: UNIVERSITY OF MIAMI, MIAMI, FLORIDA
SPONSORING ORG.: NATIONAL INSTITUTE OF GENERAL MEDICAL SCIENCES
FY : 2001
SUMMARY: The overall goal of this program is to increase our understanding of the cellular and molecular events controlling the formation of connective tissue during wound repair. The current experiments are designed to determine the physiological role of a newly identified peptide. Connective Tissue Growth Factor (CTGF). CTGF is a cysteine-rich mitogenic peptide that binds heparin and is secreted by fibroblasts after activation with Transforming Growth Factor Beta (TGF-beta). CTGF os a member of a highly conserved family of peptides that include immediate early gene products cef10, **cyr 61** , fisp 12; a putative avian proto-oncogene, nov; and a drosophila gene, twisted gastrulation, tsg, that controls medial mesoderm induction during dorsal-ventral axis pattern formation, a process also controlled by TGF-beta related peptides (dpp, scw). In the adult mammal, CTGF functions as a downstream mediator of TGF-beta1 action on connective tissue cells, where it stimulates cell proliferation and extracellular matrix synthesis. CTGF does not appear to act on epithelial cells or immune cells. Because the biological actions of TGF-beta are complex and effect many different cell types, CTGF may serve as a more specific target for selective intervention in processes involving connective tissue formation during wound repair or fibrotic disorders. Molecular and cell biological techniques will be used to produce recombinant CTGF and fragments of CTGF for investigation of its action on cells in culture and in animal models of wound repair. Specific antibodies will be produced to neutralize the biological actions of CTGF, and these will be used to determine the relationship of CTGF-function to TGF-beta's action on target cells. Agents that inhibit the induction of CTGF

expression by TGF-beta will be used for the same purpose. In situ hybridization will be used to determine the normal cell and tissue pattern of CTGF gene expression during tissue regeneration (wound repair) and embryogenesis. Transgenic mice will be generated to determine if the TGF-beta response element in the CTGF promoter is responsible for regulation of the cell and tissue specific expression of the CTGF gene. In other transgenic mice, the CTGF gene will be over-expressed in tissues prone to fibrosis, to determine if CTGF alone is sufficient to induce a fibrotic lesion. These experiments could lead to the development of new animal models for fibrotic disease and to new therapeutic approaches for the control of fibrotic disorders in humans.

15/3,AB/17 (Item 3 from file: 266)
DIALOG(R)File 266:FEDRIP

00294595
IDENTIFYING NO.: 5R01CA80080-03 AGENCY CODE: CRISP
INDUCIBLE GENES IN ANGIOGENESIS AND TUMOR GROWTH
PRINCIPAL INVESTIGATOR: LAU, LESTER F
ADDRESS: LESTER F. LAU, PHD DEPT OF MOLECULAR GENETICS CHICAGO, IL 60607-7170
PERFORMING ORG.: UNIVERSITY OF ILLINOIS AT CHICAGO, CHICAGO, ILLINOIS
SPONSORING ORG.: NATIONAL CANCER INSTITUTE
FY : 2001
SUMMARY: DESCRIPTION (Adapted from Investigator's Abstract): Angiogenesis, the sprouting of new capillaries from existing vessels, is an important process that governs embryonic development, reproduction, wound healing, and a spectrum of diseases including cancer. Recent studies have shown that angiogenesis is essential for the growth of solid tumors beyond a certain size, and intervention with tumor angiogenesis can dramatically restrict tumor development. Thus, understanding the control of angiogenesis has emerged as a challenge of fundamental biological significance, and may lead to therapeutic possibilities for the treatment of cancer. Through the characterization of growth factor induced genes, a new angiogenic inducer has been recently discovered. **Cyr61** , encoded by a growth factor inducible, immediate early gene, promotes endothelial cell adhesion, enhances growth factor induced DNA synthesis in endothelial cells, stimulates directed capillary endothelial cell migration in vitro, and induces neovascularization in vivo. **Cyr61** is a novel ligand of the integrin alphavbeta3, known to be important for angiogenesis. Furthermore, expression of **cyr61** in a tumor cell line that does not normally express it enhances the size and vascularization of tumors that develop from these cells. **Cyr61** is a member of a conserved protein family, suggesting that other members of this family may also regulate antiogenesis. Together, these findings suggest that **Cyr61** and related proteins are novel angiogenic regulators whose activities and functions merit further investigation. The activities and functions of **Cyr61** and related proteins are examined in this proposal through several approaches. First, the angiogenic activities of these proteins will be determined and the role of integrin alphavbeta3 in their actions will be assessed. Second, the structure and function relationships of **Cyr61** will be analyzed. Third, the role of **Cyr61** in tumor growth and metastasis will be evaluated, and intervention of **Cyr61** activities will be explored as a means of restricting tumor growth.

15/3,AB/18 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

122153229 CA: 122(13)153229u DISSERTATION
Regulation of expression of growth factor-inducible immediate-early genes CYR61 and PIP92
AUTHOR(S): Latinkic, Branko Vasilije
LOCATION: Univ. Illinois, Chicago, IL, USA
DATE: 1994 PAGES: 186 pp. CODEN: DABBBA LANGUAGE: English CITATION: Diss. Abstr. Int. B 1994, 55(5), 1749 AVAIL: Univ. Microfilms Int., Order No. DA9426517

15/3,AB/19 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

120185699 CA: 120(15)185699x DISSERTATION
Biochemical and functional analysis of Cyr61, the product of a growth factor-inducible immediate early gene
AUTHOR(S): Yang, George P.
LOCATION: Health Sci. Cent., Univ. Illinois, Chicago, IL, USA
DATE: 1993 PAGES: 129 pp. CODEN: DABBBA LANGUAGE: English CITATION: Diss. Abstr. Int. B 1994, 54(8), 3953 AVAIL: Univ. Microfilms Int., Order No. DA9335171

15/3,AB/20 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

120126810 CA: 120(11)126810d DISSERTATION
Characterization and expression analysis of the growth factor-inducible immediate-early gene cyr61
AUTHOR(S): O'Brien, Timothy Paul
LOCATION: Health Sci. Cent., Univ. Illinois, Chicago, IL, USA
DATE: 1992 PAGES: 126 pp. CODEN: DABBBA LANGUAGE: English CITATION: Diss. Abstr. Int. B 1993, 53(8), 3911 AVAIL: Univ. Microfilms Int., Order No. DA9238021

15/3,AB/21 (Item 1 from file: 77)
DIALOG(R)File 77:Conference Papers Index

4366040
Supplier Accession Number: 98-03872 V26N04
Extracellular matrix signaling protein CYR61 is downregulated in human prostate and kidney cancer
Froschermaier, S.E.; Schmidt, U.; Eissrich, C.; Stade, J.; Pilarsky, C.P. ; Haase, M.; Faller, G.; Kirchner, T.W.; Wirth, M.P.
European Association of Urology XIVth Congress 9810518 Stockholm (Sweden) 21-25 Mar 1998
European Association of Urology
EAU Congress Office, P.O. Box 204, 6600 AE WIJCHEN, The Netherlands; phone: +31 (0)24 6452510; fax: +31 (0)24 64550769; URL: http://www.bpc.nl/uroweb/, Abstracts available. Poster Paper No. 527
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23/3,AB/1 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

04132686 Genuine Article#: RG483 Number of References: 44
Title: EFFECTS OF FIBRIN ON THE ANGIOGENESIS IN-VITRO OF BOVINE ENDOTHELIAL-CELLS IN COLLAGEN GEL (Abstract Available)
Author(s): TAKEI A; TASHIRO Y; NAKASHIMA Y; SUEISHI K
Corporate Source: KYUSHU UNIV 60,FAC MED,DEPT PATHOL 1,HIGASHI KU,3-1-1 MAIDASHI/FUKUOKA 812//JAPAN/
Journal: IN VITRO CELLULAR & DEVELOPMENTAL BIOLOGY-ANIMAL, 1995, V31, N6 (JUN), P467-472
ISSN: 1071-2690
Language: ENGLISH Document Type: ARTICLE
Abstract: The effect of fibrin on angiogenesis in vitro was investigated using an experimental model of tube formation by bovine capillary endothelial cells (BCEs) in type I collagen **gel** . One milligram per milliliter of fibrin added into type I collagen **gel** significantly increased the length of the tubular structures formed by BCEs in the **gel** by about 180% compared with type I collagen only. The facilitating effect of fibrin on tube formation by BCEs was inhibited by either anti-basic **fibroblast** growth factor (bFGF) IgG (25 mu g/ml) or anti-urokinase type plasminogen activator (uPA) IgG (10 mu g/ml) added to the **gel** and culture medium, but not by anti-tissue type plasminogen activator (10 mu g/ml) or non-immune IgG. The Arg-Gly-Asp (RGD) containing peptides (100 mu g/ml) added to the culture medium also suppressed tube formation by BCEs in fibrin-containing type I collagen **gel** , but not in type I collagen **gel** . These results suggest that the increased release of bFGF and uPA by BCEs therefore plays a role in the angiogenic effect of fibrin in vitro, and the angiogenic effect of fibrin is mediated by the RGD sequence in fibrin, probably via the function of **integrin** receptor of the BCEs.

23/3,AB/18 (Item 15 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00318172
A METHOD OF SCREENING FOR INHIBITORS OF HEPARIN-BINDING PROTEIN
PROCEDE DE DEPISTAGE D'INHIBITEURS DE LA PROTEINE FIXATRICE D'HEPARINE
Patent Applicant/Assignee:
NOVO NORDISK AS
FLODGAARD Hans
Inventor(s):
FLODGAARD Hans
Patent and Priority Information (Country, Number, Date):
Patent: WO 9305396 A1 19930318
Application: WO 92DK270 19920909 (PCT/WO DK9200270)
Priority Application: WO 91DK264 19910912
Designated States: AU CA CS FI HU JP PL RU US AT BE CH DE DK ES FR GB GR IE IT LU MC NL SE
Publication Language: English
Fulltext Word Count: 6245

English Abstract
Inhibitors of heparin-binding protein are screened for by incubating HBP or a cell producing HBP with a substance suspected of being an HBP inhibitor and with tissue, cells or a component thereof capable of interacting with HBP, and detecting any effect of said substance on the

interaction of HBP with said tissue, cells or component thereof, decreased interaction indicating that said substance is an HBP inhibitor.

Japanese Abstract
Le depistage d'inhibiteurs de la proteine fixatrice d'heparine (HBP) s'effectue par l'incubation de la HBP ou d'une cellule produisant la HBP avec une substance dont on pense qu'elle est une inhibitrice de HBP, ainsi qu'avec des tissus, des cellules ou un constituant de ceux-ci qui est (sont) apte(s) a interagir avec la HBP; et par la detection de tout effet qu'aurait ladite substance sur l'interaction entre la HBP et les tissus, les cellules ou le constituant de ceux-ci. Toute diminution de l'interaction indiquerait que ladite substance est un inhibiteur de HBP.

23/3,AB/19 (Item 16 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00311634
INHIBITION OF SMOOTH MUSCLE CELL PROLIFERATION BY VITRONECTIN
INHIBITION DE PROLIFERATION DE CELLULES MUSCULAIRES LISSES PAR LA VITRONECTINE
Patent Applicant/Assignee:
THROMBOSIS RESEARCH INSTITUTE
MAX PLANCK GESELLSCHAFT
WIJELATH Errol
DEMOLIOU-MASON Catherine
HESS Sibylle
PREISSNER Klaus Theodore
KAKKAR Vijay Vir
Inventor(s):
WIJELATH Errol
DEMOLIOU-MASON Catherine
HESS Sibylle
PREISSNER Klaus Theodore
KAKKAR Vijay Vir
Patent and Priority Information (Country, Number, Date):
Patent: WO 9221363 A1 19921210
Application: WO 92GB958 19920527 (PCT/WO GB9200958)
Priority Application: GB 9111439 19910528
Designated States: AT AT AU BB BE BF BG BJ BR CA CF CG CH CH CI CM CS DE DE DK DK ES ES FI GB GB GN GR HU IT JP KP KR LK LU LU MC MG ML MN MR MW NL NL NO PL RO RU SE SN TD TG US
Publication Language: English
Fulltext Word Count: 4228

English Abstract
Vitronectin, an adhesion protein, acts as a growth inhibitory modulator of vascular smooth muscle cell proliferation, a phenomenon which is known to cause atherosclerosis. The invention provides a method of inhibiting smooth muscle cell proliferation comprising administration of vitronectin, as well as vitronectin for such use and pharmaceutical compositions containing vitronectin. Preferably, the vitronectin is the extended form thereof.

Japanese Abstract
La vitronectine, une proteine d'adhesion, agit comme un modulateur inhibiteur de croissance par rapport a la proliferation de cellules musculaires lisses, un phenomene dont on sait qu'il provoque l'atherosclerose. L'invention se rapporte a un procede d'inhibition de la proliferation de cellules musculaires lisses, consistant a administrer de

la vitronectine, ainsi qu'a la vitronectine utilisee a de telles fins et des compositions pharmaceutiques contenant cette proteine. De preference, la vitronectine est sous sa forme allongee.

23/3,AB/25 (Item 1 from file: 654)
DIALOG(R)File 654:US PAT.FULL.

02581827

Utility
USE OF ISOLATED DOMAINS OF TYPE IV COLLAGEN TO MODIFY CELL AND TISSUE INTERACTIONS
[Antigrowth agents]

PATENT NO.: 5,567,609
ISSUED: October 22, 1996 (19961022)
INVENTOR(s): Sarras, Jr. Michael P., Kansas City, KS (Kansas), US (United States of America)
Hudson, Billy G., Lenexa, KS (Kansas), US (United States of America)
ASSIGNEE(s): University of Kansas Medical Center, (A U.S. Company or Corporation), Kansas City, KS (Kansas), US (United States of America)
[Assignee Code(s): 36361]
APPL. NO.: 8-268,969
FILED: June 30, 1994 (19940630)

STATEMENT OF RIGHTS

The U.S. Government has a paid-up license in this invention and the right in limited circumstances to require the patent owner to license others on reasonable terms as provided for by the terms of Grants No. 01-RR06500 and AM 18381 awarded by the National Institute of Health.

FULL TEXT: 560 lines

ABSTRACT

The instant invention demonstrates that the 7S and NC1 domains of type IV collagen disrupts cell aggregation and tissue development. Structural changes in mesoglea, inhibition of cell proliferation, and changes in cell differentiation patterns accompanies the blockage of cell aggregates which indicate that blockage may be due to alterations in mesoglea (extracellular matrix) structure with accompanying effects on cell behavior. Type IV collagen has a critical role in the initial formation of mesoglea and that perturbation of mesoglea formation affects cell division, cell differentiation, and morphogenesis.

23/3,AB/27 (Item 3 from file: 654)
DIALOG(R)File 654:US PAT.FULL.

02492522

Utility
METHOD AND SYSTEM FOR MEASUREMENT OF MECHANICAL PROPERTIES OF MOLECULES AND CELLS

PATENT NO.: 5,486,457

ISSUED: January 23, 1996 (19960123)
INVENTOR(s): Butler, James P., Brookline, MA (Massachusettes), US (United States of America)
Fredberg, Jeffrey J., Sharon, MA (Massachusettes), US (United States of America)
Ingber, Donald E., Boston, MA (Massachusettes), US (United States of America)
Wang, Ning, Brookline, MA (Massachusettes), US (United States of America)
ASSIGNEE(s): Children's Medical Center Corporation, (A U.S. Company or Corporation), Boston, MA (Massachusetts), US (United States of America)
President and Fellows of Harvard College, (A U.S. Company or Corporation), Cambridge, MA (Massachusetts), US (United States of America)
[Assignee Code(s): 542; 10709]
APPL. NO.: 8-112,757
FILED: August 25, 1993 (19930825)

BACKGROUND OF THE INVENTION

The United States government has rights in this invention by virtue of NASA grant No. NAG-9-430 and NIH grant Nos. CA4554B to Donald Ingber, NIH grant No. HL33009 to Jeffrey J. Fredberg and NIH grant No. HL36427 to James P. Butler.

FULL TEXT: 940 lines

ABSTRACT

Mechanical stresses and deformations are applied directly to cell surface receptors or molecules and measured using a system including a magnetic twisting device in combination with ferromagnetic microbeads coated with ligands for **integrins** or any other surface receptors. The system can be used diagnostically to characterize cells and molecules and to determine the effect of transformation and compounds, including drugs, on the cells and molecules. The system can also be used to induce cells to grow or alter production of molecules by the cells.
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25/3,AB/2 (Item 1 fr m file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05820700 Genuine Article#: WZ851 Number of References: 88
Title: Heparan sulfate **proteoglycans function in the binding and degradation of vitronectin by** fibroblast **monolayers** (ABSTRACT AVAILABLE)
Author(s): WilkinsPort CE; McKeownLongo PJ (REPRINT)
Corporate Source: ALBANY MED COLL UNION UNIV,ALBANY MED COLL, DEPT PHYSIOL & CELL BIOL, NEIL HELLMAN MED RES BLDG/ALBANY//NY/12208 (REPRINT); ALBANY MED COLL UNION UNIV,ALBANY MED COLL, DEPT PHYSIOL & CELL BIOL/ALBANY//NY/12208
Journal: BIOCHEMISTRY AND CELL BIOLOGY-BIOCHIMIE ET BIOLOGIE CELLULAIRE, 1996, V74, N6, P887-897
ISSN: 0829-8211 Publication date: 19960000
Publisher: NATL RESEARCH COUNCIL CANADA, RESEARCH JOURNALS, MONTREAL RD, OTTAWA ON K1A 0R6, CANADA
Language: English Document Type: ARTICLE
Abstract: Vitronectin, a 75-kDa plasma protein is also found in the extracellular matrix, where it is believed to promote cell adhesion and **migration** . In addition to its role in adhesion, matrix vitronectin is also believed to function as an opsonin promoting the clearance of thrombin-serpin complexes from the matrix. Vitronectin is cleared from the matrix by receptor-mediated endocytosis followed by lysosomal degradation, suggesting that cells can regulate the levels of vitronectin present in the matrix. However, the mechanism by which plasma vitronectin associates with the extracellular matrix remains unclear. Studies were conducted to define the binding site(s) for vitronectin in **fibroblast** cell layers. Sodium chlorate, a competitive inhibitor of proteoglycan sulfation, produced a dose-dependent decrease in both binding and degradation of vitronectin. This inhibition was reversible in that removal of chlorate returned both binding and degradation of vitronectin to near control levels within 24 h. The binding of vitronectin to cell layers was not dependent on cells because vitronectin bound directly to isolated matrix. Isolated matrices prepared from cell layers treated with sodium chlorate also exhibited a dose-dependent decrease in vitronectin binding, consistent with the binding site for vitronectin in the matrix being sulfated proteoglycans. Binding and degradation of vitronectin were also sensitive to the addition of exogenous heparin, suggesting that the heparin binding domain of vitronectin was mediating binding to the matrix. Incubating **fibroblast** monolayers with heparinase III resulted in a 40% decrease in binding and degradation of vitronectin. Taken together, the above findings suggest that vitronectin's binding to the matrix and its subsequent degradation are dependent on **heparan sulfate** proteoglycans.

25/3,AB/4 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05356347 Genuine Article#: VT694 Number of References: 50
Title: HEPARAN- SULFATE **PROTEOGLYCAN ON LEUKEMIC-CELLS IS PRIMARILY INVOLVED IN** INTEGRIN **TRIGGERING AND ITS MEDIATED ADHESION TO ENDOTHELIAL-CELLS** (Abstract Available)
Author(s): TANAKA Y; KIMATA K; WAKE A; MINE S; MORIMOTO I; YAMAKAWA N; HABUCHI H; ASHIKARI S; YAMAMOTO H; SAKURAI K; YOSHIDA K; SUZUKI S; ETO S
Corporate Source: UNIV OCCUPAT & ENVIRONM HLTH,SCH MED,DEPT INTERNAL MED

1,YAHATANISHI KU,1-1 ISEIGAOKA/KITAKYUSHU/FUKUOKA 807/JAPAN/; AICHI MED UNIV,INST MOL SCI MED/NAGAKUTE/AICHI 48011/JAPAN/; SEIKAGAKU KOGYO CO LTD,TOKYO RES INST/HIGASHI YAMATO 207//JAPAN/
Journal: JOURNAL OF EXPERIMENTAL MEDICINE, 1996, V184, N5 (NOV 1), P 1987-1997
ISSN: 0022-1007
Language: ENGLISH Document Type: ARTICLE
Abstract: Leukocyte **migration** from circulation into tissue depends on leukocyte **integrin** -mediated adhesion to endothelium, but **integrins** cannot function until activated. However, it remains to be understood how tumor cells adhere to endothelium and infiltrate into underlying tissue. We studied mechanisms of extravasation of leukemic cells using adult T cell leukemia (ATL) cells and report the following novel features of cell surface **heparan sulfate** proteoglycan on ATL cells in ATL cell adhesion to endothelium:ATL cells adhere to endothelial cells through already activated **integrins** without exogenous stimulation; different from any other hematopoietic cells, ATL cells express a characteristic **heparan sulfate** capable of immobilizing heparin-binding chemokine macrophage inflammatory protein (MIP)-1 beta, a potent T cell **integrin** trigger, produced by the cells themselves; competitive interruption of endogenous **heparan sulfate** proteoglycan synthesis reduces cell surface MIP-1 beta and prevents ATL cells from **integrin** -mediated adhesion to endothelial cells or intercellular adhesion molecule-1 triggered through G-protein. We propose that leukemic cells adhere to endothelial cells through the adhesion cascade, similar to normal leukocyte, and that the cell surface **heparan sulfate** , particularly on ATL cells, is pivotally involved in chemokine-dependent autocrine stimulation of **integrin** triggering by immobilizing the chemokine on them.

25/3,AB/8 (Item 7 from file: 34)

04469281 Genuine Article#: TF223 Number of References: 1289
Title: CELL-ADHESION MOLECULES .1. IMMUNOGLOBULIN SUPERFAMILY
Author(s): BRUMMENDORF T; RATHJEN FG
Corporate Source: MAX PLANCK INST ENTWICKLUNGSBIOL,SPEMANNSTR 35/D-72076 TUBINGEN//GERMANY/; MAX DELBRUCK CENTRUM MOLEK MED/D-13122 BERLIN//GERMANY/
Journal: PROTEIN PROFILE, 1995, V2, N9, P963&
ISSN: 1070-3667
Language: ENGLISH Document Type: REVIEW

25/3,AB/35 (Item 34 from file: 34)

01812252 Genuine Article#: JC512 Number of References: 84
Title: A CELL-SURFACE HEPARAN- SULFATE **PROTEOGLYCAN MEDIATES NEURAL CELL-ADHESION AND SPREADING ON A DEFINED SEQUENCE FROM THE C-TERMINAL CELL AND HEPARIN BINDING DOMAIN OF FIBRONECTIN, FN-C/H-II** (Abstract Available)
Author(s): HAUGEN PK; LETOURNEAU PC; DRAKE SL; FURCHT LT; MCCARTHY JB
Corporate Source: WASHINGTON UNIV,SCH MED,DEPT PATHOL & MED,BOX 8118,660 S EUCLID AVE/ST LOUIS//MO/63110; UNIV MINNESOTA,DEPT CELL BIOL & ANAT/MINNEAPOLIS//MN/55455; UNIV MINNESOTA,DEPT LAB MED & PATHOL/MINNEAPOLIS//MN/55455; UNIV MINNESOTA,CTR BIOMED ENGN/MINNEAPOLIS//MN/55455

Journal: JOURNAL OF NEUROSCIENCE, 1992, V12, N7 (JUL), P2597-2608
Language: ENGLISH Document Type: ARTICLE
Abstract: FN-C/H II is a heparin binding synthetic peptide from the C-terminal cell and heparin binding domain of fibronectin (FN) that mediates neuronal cell adhesion, spreading, and neurite outgrowth. Cellular interactions with FN-C/H II are inhibited by soluble heparin, suggesting that a cell-surface proteoglycan may mediate interactions with FN-C/H II (Haugen et al., 1990). To test this hypothesis further, **heparan sulfate** (HS) or chondroitin sulfate (CS) was removed from the cell surface by enzyme treatment. Heparitinase but not chondroitinase treatment of cells inhibited rat B104 neuroblastoma cell adhesion and spreading on FN-C/H II. Additionally, heparitinase treatment decreased the spreading of cells on the 33/66 kDa fragments containing the C-terminal heparin binding domain of FN. Furthermore, antibodies generated against a mouse melanoma HS proteoglycan (HSPG) inhibited B104 cell adhesion to FN-C/H II and the 33/66 kDa FN fragments. S-35-HSPG isolated from B104 cells directly bound to FN-C/H II both in solid phase assays and by affinity chromatography, but failed to bind to a control peptide from this region, CS1. The binding of S-35-HSPG was predominantly mediated by the HS and not the core protein of the HSPG. SDS-PAGE of iodinated HSPG demonstrated a single 78 kDa core protein following heparitinase digestion, which **migrated** at 51 kDa under nonreducing conditions. Anti-HSPG antibodies recognized the 78 kDa core protein by immunoblotting, and stained the surface of rat B104 neuroblastoma cells and cells of the primary neonatal rat nervous system. These results identify a cell-surface HSPG that likely mediates neuronal cell binding interactions with FN-C/H II.
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29/3,AB/1 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

05411985 Genuine Article#: VW971 Number of References: 76
Title: N-SYNDECAN - STRUCTURE AND FUNCTION OF A TRANSMEMBRANE HEPARAN-SULFATE **PROTEOGLYCAN** (Abstract Available)
Author(s): CAREY DJ
Corporate Source: WEIS CTR RES,GEISINGER CLIN/DANVILLE//PA/17822
Journal: PERSPECTIVES ON DEVELOPMENTAL NEUROBIOLOGY, 1996, V3, N4, P331-346
ISSN: 1064-0517
Language: ENGLISH Document Type: ARTICLE
Abstract: N-syndecan is a member of the syndecan family of transmembrane **heparan sulfate** proteoglycans that was cloned initially from neonatal rat Schwann cells and is the principal syndecan expressed during early postnatal development in the central and peripheral nervous systems. Purified N-syndecan binds in vitro with high affinity to several extracellular regulatory ligands, including basic **fibroblast** growth factor, the secreted adhesive protein heparin binding growth-associated molecule, and a novel **collagen** -like protein secreted by Schwann cells. These extracellular ligands utilize the **heparan sulfate** chains of N-syndecan for binding. Based on the striking amino acid sequence homology of the cytoplasmic domain of N-syndecan to syndecan-1, it is proposed that N-syndecan associates with the actin-based cytoskeleton. N-syndecan core proteins self associate by means of an unusual dimerization motif comprised of the transmembrane domain and a short flanking sequence in the ectodomain. Similar to other single transmembrane domain receptor proteins, it is suggested that ligand-regulated dimerization of N-syndecan represents a mechanism for regulating downstream signaling activities. In rat brain tissue a significant fraction of the N-syndecan molecules are present in a soluble form, presumably as a result of proteolytic membrane shedding. A model is presented for morphoregulatory activity of N-syndecan in which extracellular ligand-induced clustering of N-syndecan molecules on the cell surface promotes cytoskeletal association and reorganization. Membrane shedding separates the functional domains of the proteoglycan and terminates this activity.

29/3,AB/3 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

04132686 Genuine Article#: RG483 Number of References: 44
Title: EFFECTS OF FIBRIN **ON THE ANGIOGENESIS IN-VITRO OF BOVINE ENDOTHELIAL-CELLS IN** COLLAGEN **GEL** (Abstract Available)
Author(s): TAKEI A; TASHIRO Y; NAKASHIMA Y; SUEISHI K
Corporate Source: KYUSHU UNIV 60,FAC MED,DEPT PATHOL 1,HIGASHI KU,3-1-1 MAIDASHI/FUKUOKA 812//JAPAN/
Journal: IN VITRO CELLULAR & DEVELOPMENTAL BIOLOGY-ANIMAL, 1995, V31, N6 (JUN), P467-472
ISSN: 1071-2690
Language: ENGLISH Document Type: ARTICLE
Abstract: The effect of **fibrin** on angiogenesis in vitro was investigated using an experimental model of tube formation by bovine capillary endothelial cells (BCEs) in type I **collagen** gel. One milligram per milliliter of **fibrin** added into type I **collagen** gel significantly increased the length of the tubular structures formed by BCEs in the gel by about 180% compared with type I **collagen** only. The facilitating effect of **fibrin** on tube formation by BCEs was inhibited

by either anti-basic **fibroblast** growth factor (bFGF) IgG (25 mu g/ml) or anti-urokinase type plasminogen activator (uPA) IgG (10 mu g/ml) added to the gel and culture medium, but not by anti-tissue type plasminogen activator (10 mu g/ml) or non-immune IgG. The Arg-Gly-Asp (RGD) containing peptides (100 mu g/ml) added to the culture medium also suppressed tube formation by BCEs in **fibrin** -containing type I **collagen** gel, but not in type I **collagen** gel. These results suggest that the increased release of bFGF and uPA by BCEs therefore plays a role in the angiogenic effect of **fibrin** in vitro, and the angiogenic effect of **fibrin** is mediated by the RGD sequence in **fibrin** , probably via the function of **integrin** receptor of the BCEs.

29/3,AB/4 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

04010749 Genuine Article#: QZ091 Number of References: 76
Title: LOSS OF CELL-SURFACE SYNDECAN-1 CAUSES EPITHELIA TO TRANSFORM INTO ANCHORAGE-INDEPENDENT MESENCHYME-LIKE CELLS (Abstract Available)
Author(s): KATO M; SAUNDERS S; NGUYEN H; BERNFIELD M
Corporate Source: HARVARD UNIV,SCH MED,JOINT PROGRAM NEONATOL,300LONGWOOD AVE/BOSTON//MA/02115; HARVARD UNIV,SCH MED,JOINT PROGRAM NEONATOL/BOSTON//MA/02115
Journal: MOLECULAR BIOLOGY OF THE CELL, 1995, V6, N5 (MAY), P559-576
ISSN: 1059-1524
Language: ENGLISH Document Type: ARTICLE
Abstract: Simple epithelial cells are polygonal in shape, polarized in an apical-basal orientation, and organized into closely adherent sheets, characteristics that result from a variety of cellular specializations and adhesive proteins. These characteristics are lost when the epithelia transform during embryogenesis into mesenchymal cells or after neoplasia into invasive carcinoma cells. Of the syndecan family of transmembrane **heparan sulfate** proteoglycans, simple epithelia produce predominately syndecan-1, which is found at basolateral surfaces and within adhesive junctions. To elucidate the function of this syndecan-1, normal murine mammary gland epithelia were made deficient in syndecan-1 by transfection with an expression vector containing the syndecan-1 cDNA in the antisense configuration. Several independently derived clones of stable transfectants contained the antisense cDNA in their genome and expressed the antisense transcript. These grew either as epithelial islands of closely adherent polygonal cells, identical to both the parental cells and the vector-only control transfectants, or as individual elongated fusiform cells that invaded and **migrated** within **collagen** gels, like mesenchymal cells, but were anchorage-independent for growth. The clones that retained epithelial characteristics were moderately deficient in cell surface syndecan-1 (greater than 48% of control levels) but did not differ from control cells in expression of beta 1-**integrins** and E-cadherin, or in F-actin organization. However, the clones of fusiform cells were severely deficient in cell surface syndecan-1 (less than 12% of control levels) and showed rearranged beta 1-**integrins** , markedly reduced E-cadherin expression, and disorganized F-actin filaments, but retained mammary epithelial markers. Therefore, depleting epithelia of cell surface syndecan-1 alters cell morphology and organization, the arrangement and expression of adhesion molecules, and anchorage-dependent growth controls. Thus, cell surface syndecan-1 is required to maintain the normal phenotype of simple epithelia.

29/3,AB/6 (Item 6 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03729178 Genuine Article#: QA850 Number of References: 30
Title: THE ROLE OF CYTOKINES AND EXTRACELLULAR-MATRIX PROTEINS FOR THE GASTRIC-ULCER HEALING-PROCESS (Abstract Available)
Author(s): GILLESSEN A; DOMSCHKE W
Corporate Source: UNIV MUNSTER,MED KLIN B,ALBERT SCHWEITZER STR 33/D-48129 MUNSTER//GERMANY/; UNIV MUNSTER,MED KLIN & POLIKLIN B/MUNSTER//GERMANY/
Journal: ZEITSCHRIFT FUR GASTROENTEROLOGIE, 1994, V32, N12 (DEC), P691-693
ISSN: 0044-2771
Language: GERMAN Document Type: REVIEW
Abstract: The healing process of gastric ulcers is regulated by many factors. Cytokines play a central role in the different stages of healing. in the early stage of inflammation, TNF-alpha and interleukins regulate cell-**migration** and -proliferation. In the ulcer's base revascularisation is stimulated by b-FGF, **collagen** synthesis by TGF-beta. The reepithelialization is mediated by EGF, which accelerates ulcer healing in animal models; while TGF-alpha is involved in mucosal protection.

The extracellular matrix (ECM) is important for the stability and quality of the ulcer scar, as known from dermatological experiments. An increase of **collagen** types I and III in healing gastric ulcers was demonstrated recently, which might illuminate, the particular role of ECM proteins for the gastric ulcer healing process.

29/3,AB/7 (Item 7 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03402834 Genuine Article#: PC885 Number of References: 65
Title: THE ROLE OF EXTRACELLULAR-MATRIX IN POSTINFLAMMATORY WOUND-HEALING AND FIBROSIS (Abstract Available)
Author(s): RAGHOW R
Corporate Source: UNIV TENNESSEE,COLL MED,DEPT PHARMACOL/MEMPHIS//TN/38104; VET AFFAIRS MED CTR/MEMPHIS//TN/38104
Journal: FASEB JOURNAL, 1994, V8, N11 (AUG), P823-831
ISSN: 0892-6638
Language: ENGLISH Document Type: REVIEW
Abstract: Massive cell **migration** , proliferation, phenotypic differentiation, and enhanced biosynthetic activities characterize the sites of wound healing and fibrosis. Regulation of cellular functions by extracellular matrix, which consists of a dynamic assemblage of a variety of interacting molecules capable of reorganization in response to endogenous and exogenous stimuli, represents a fundamental epigenetic mechanism regulating cellular behavior and phenotype. Interactions of the individual components of extracellular matrix with specific cell surface molecules, **integrin** receptors, and proteoglycans initiate a cascade of signal transduction leading to varied short-term or persistent cellular responses. Extracellular matrix also serves as an important reservoir of cytokines and growth factors, thus modulating the action of a host of potent biological response modifiers by their selective, local accumulation and release. Currently known mechanisms by which extracellular matrix modulates different facets of the process of tissue remodeling after injury, which culminate either in normal wound repair or fibrosis, are discussed.

29/3,AB/8 (Item 8 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03355493 Genuine Article#: PA126 Number of References: 51
Title: EXPRESSION OF SYNDECAN-1 INHIBITS CELL INVASION INTO TYPE-I COLLAGEN (Abstract Available)
Author(s): LIEBERSBACH BF; SANDERSON RD
Corporate Source: UNIV ARKANSAS MED SCI HOSP,DEPT PATHOL SLOT 517,4301 W MARKHAM/LITTLE ROCK//AR/72205; UNIV ARKANSAS MED SCI HOSP,DEPT PATHOL SLOT 517/LITTLE ROCK//AR/72205; UNIV ARKANSAS MED SCI HOSP,DEPT ANAT/LITTLE ROCK//AR/72205; UNIV ARKANSAS MED SCI HOSP,MCCLURE MUSCULOSKELETAL RES CTR/LITTLE ROCK//AR/72205
Journal: JOURNAL OF BIOLOGICAL CHEMISTRY, 1994, V269, N31 (AUG 5), P 20013-20019
ISSN: 0021-9258
Language: ENGLISH Document Type: ARTICLE
Abstract: Before cells can invade the extracellular matrix, they must alter their expression of adhesion receptors. Syndecan-1 is a cell surface proteoglycan that binds cells to matrix and undergoes changes in expression during development, during differentiation of some cell types, and following malignant transformation in some tumors. To determine if changes in syndecan-1 expression influence cell invasion, we employed a model system in which human B lymphoid cells invade type I **collagen** gels. Examination of a panel of cell lines reveals that those lines not expressing syndecan-1 invade and **migrate** within **collagen** . In contrast, cell lines expressing syndecan-1 fail to invade. To directly assess the effect of syndecan-1 on invasion, ARH-77 cells, which do not express syndecan-1 and readily invade **collagen** , were transfected with a cDNA for syndecan-1. The syndecan-1-positive transfectants exhibit a drastically reduced ability to invade as compared to parental cells. This inhibition of invasion by syndecan-1 is reversed by preincubating gels with heparin or by growing cells in chlorate, an inhibitor of glycosaminoglycan sulfation. These results demonstrate that expression of syndecan-1 inhibits cell invasion into **collagen** and that loss of syndecan-1 expression may be necessary prior to the **migration** of normal or metastatic cells.

29/3,AB/9 (Item 9 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03239711 Genuine Article#: NP417 Number of References: 33
Title: INTERACTION OF INTERLEUKIN-1 AND INTERFERON-GAMMA ON FIBROBLAST GROWTH FACTOR-INDUCED ANGIOGENESIS (Abstract Available)
Author(s): NORIOKA K; MITAKA T; MOCHIZUKI Y; HARA M; KAWAGOE M; NAKAMURA H
Corporate Source: SAPPORO MED UNIV,SCH MED,CANC RES INST,DEPT PATHOL,CHUO KU,SOUTH 1,WEST 17/SAPPORO/HOKKAIDO 060/JAPAN/; NATL DEF MED COLL,DEPT INTERNAL MED 1/TOKOROZAWA/SAITAMA 359/JAPAN/
Journal: JAPANESE JOURNAL OF CANCER RESEARCH, 1994, V85, N5 (MAY), P522-529
ISSN: 0910-5050
Language: ENGLISH Document Type: ARTICLE
Abstract: The interaction of interleukin-1 (IL-1) and interferon-gamma (IFN-gamma) actions on several aspects of angiogenesis in vitro and in vivo was studied. The proliferation and **migration** of human umbilical vein endothelial cells cultured with basic **fibroblast** growth factor (bFGF) were synergistically inhibited by cotreatment with IL-1 and IFN-gamma. Endothelial cell adhesion to **collagen** was suppressed by IL-1 and the effect was slightly enhanced by the combination of IL-1 and IFN-gamma. Local administration of IL-I (10,000 U) and IFN-gamma

(1,000 U) inhibited bFGF-induced angiogenesis in the skin of mice, and synergistic inhibitory activity of the combination was demonstrated. Expression of FGF receptors was strongly downregulated by the combination, whereas expressions of epidermal growth factor (EGF) receptors, **integrin** beta(1) and **integrin** beta(3) were not. EGF partially abrogated the growth-inhibitory effects of IL-1 and IFN-gamma. These findings indicate that IL-1 and IFN-gamma are each able to act an angiogenesis inhibitor in a situation where FGF plays a major role in angiogenesis, and the activity is synergistically enhanced when they are used in combination.

29/3,AB/10 (Item 10 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

03223800 Genuine Article#: NN473 Number of References: 56
Title: MAPPING THE HEPARIN-BINDING SITES ON TYPE-I COLLAGEN **MONOMERS AND FIBRILS** (Abstract Available)
Author(s): SANANTONIO JD; LANDER AD; KARNOVSKY MJ; SLAYTER HS
Corporate Source: HARVARD UNIV,SCH MED,DANA FARBER CANC INST,MOLEC STRUCT LAB,44 BINNEY ST,JFBG10/BOSTON//MA/02115; HARVARD UNIV,SCH MED,DANA FARBER CANC INST,MOLEC STRUCT LAB/BOSTON//MA/02115; HARVARD UNIV,SCH MED,DEPT PATHOL/BOSTON//MA/02115; MIT,DEPT BRAIN & COGNIT SCI/CAMBRIDGE//MA/02139; MIT,DEPT BIOL/CAMBRIDGE//MA/02139; HARVARD UNIV,SCH MED,DEPT CELLULAR & MOLEC PHYSIOL/BOSTON//MA/02115
Journal: JOURNAL OF CELL BIOLOGY, 1994, V125, N5 (JUN), P1179-1188
ISSN: 0021-9525
Language: ENGLISH Document Type: ARTICLE
Abstract: The glycosaminoglycan chains of cell surface **heparan sulfate** proteoglycans are believed to regulate cell adhesion, proliferation, and extracellular matrix assembly, through their interactions with heparin-binding proteins (for review see Ruoslahti, E. 1988. Annu. Rev Cell Biol. 4:229-255; and Bernfield, M., R. Kokenyesi, M. Kato, M. T. Hinkes, J. Spring, R. L. Gallo, and E. J. Lose. 1992. Annu. Rev. Cell Biol. 8:365-393). Heparin-binding sites on many extracellular matrix proteins have been described; however, the heparin-binding site on type I **collagen** , a ubiquitous heparin-binding protein of the extracellular matrix, remains undescribed. Here we used heparin, a structural and functional analogue of **heparan sulfate** , as a probe to study the nature of the **heparan sulfate** proteoglycan-binding site on type I **collagen** . We used affinity coelectrophoresis to study the binding of heparin to various forms of type I **collagen** , and electron microscopy to visualize the site(s) of interaction of heparin with type I **collagen** monomers and fibrils. Using affinity coelectrophoresis it was found that heparin has similar affinities for both procollagen and **collagen** fibrils (K-d's similar to 60-80 nM), suggesting that functionally similar heparin-binding sites exist in type I **collagen** independent of its aggregation state. Complexes of heparin-albumin-gold particles and procollagen were visualized by rotary shadowing and electron microscopy, and a preferred site of heparin binding was observed near the NH2 terminus of procollagen. Native or reconstituted type I **collagen** fibrils showed one region of significant heparin-gold binding within each 67-nm period, present near the division between the overlap and gap zones, within the ''a'' bands region. According to an accepted model of **collagen** fibril structure, our data are consistent with the presence of a single preferred heparin-binding site near the NH,, terminus of the **collagen** monomer. Correlating these data with known type I **collagen** sequences, we suggest that the heparin-binding site in type I **collagen** may consist of a highly basic triple helical domain, including several amino acids known sometimes to function as

disaccharide acceptor sites. We propose that the heparin-binding site of type I **collagen** may play a key role in cell adhesion and **migration** within connective tissues, or in the cell-directed assembly or restructuring of the collagenous extracellular matrix.

29/3,AB/12 (Item 12 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

01447851 Genuine Article#: GZ963 Number of References: 67
Title: A CELL-SURFACE CHONDROITIN SULFATE PROTEOGLYCAN, IMMUNOLOGICALLY RELATED TO CD44, IS INVOLVED IN TYPE-I COLLAGEN**-MEDIATED MELANOMA CELL MOTILITY AND INVASION** (Abstract Available)
Author(s): FAASSEN AE; SCHRAGER JA; KLEIN DJ; OEGEMA TR; COUCHMAN JR; MCCARTHY JB
Corporate Source: UNIV MINNESOTA,DEPT LAB MED & PATHOL/MINNEAPOLIS//MN/55455; UNIV MINNESOTA,DEPT PEDIAT/MINNEAPOLIS//MN/55455; UNIV MINNESOTA,DEPT ORTHOPAED SURG & BIOCHEM/MINNEAPOLIS//MN/55455; UNIV MINNESOTA,CTR BIOMED ENGN/MINNEAPOLIS//MN/55455; UNIV ALABAMA,DEPT CELL BIOL & ANAT/BIRMINGHAM//AL/35294
Journal: JOURNAL OF CELL BIOLOGY, 1992, V116, N2 (JAN), P521-531
Language: ENGLISH Document Type: ARTICLE
Abstract: The metastatic spread of tumor cells occurs through a complex series of events, one of which involves the adhesion of tumor cells to extracellular matrix (ECM) components. Multiple interactions between cell surface receptors of an adherent tumor cell and the surrounding ECM contribute to cell motility and invasion. The current studies evaluate the role of a cell surface chondroitin sulfate proteoglycan (CSPG) in the adhesion, motility, and invasive behavior of a highly metastatic mouse melanoma cell line (K1735 M4) on type I **collagen** matrices. By blocking mouse melanoma cell production of CSPG with p-nitrophenyl beta-D-xylo-pyranoside (beta-D-xyloside), a compound that uncouples chondroitin sulfate from CSPG core protein synthesis, we observed a corresponding decrease in melanoma cell motility on type I **collagen** and invasive behavior into type I **collagen** gels. Melanoma cell motility on type I **collagen** could also be inhibited by removing cell surface chondroitin sulfate with chondroitinase. In contrast, type I **collagen** -mediated melanoma cell adhesion and spreading were not affected by either beta-D-xyloside or chondroitinase treatments. These results suggest that mouse melanoma CSPG is not a primary cell adhesion receptor, but may play a role in melanoma cell motility and invasion at the level of cellular translocation. Furthermore, purified mouse melanoma cell surface CSPG was shown, by affinity chromatography and in solid phase binding assays, to bind to type I **collagen** and this interaction was shown to be mediated, at least in part, by chondroitin sulfate. Additionally we have determined that mouse melanoma CSPG is composed of a 110-kD core protein that is recognized by anti-CD44 antibodies on Western blots. Collectively, our data suggests that interactions between a cell surface CD44-related CSPG and type I **collagen** in the ECM may play an important role in mouse melanoma cell motility and invasion, and that the chondroitin sulfate portion of the proteoglycan seems to be a critical component in mediating this effect.

29/3,AB/32 (Item 17 from file: 349)
DIALOG(R)File 349:PCT Fulltext

00290410

PROCESS FOR CONTROLLING CELL GROWTH ON SURFACES
PROCEDE DE REGULATION DE LA CROISSANCE CELLULAIRE SUR DES SURFACES
Patent Applicant/Assignee:
CASE WESTERN RESERVE UNIVERSITY
Inventor(s):
SUKENIKK Chaim N
CULP Lloyd A
Patent and Priority Information (Country, Number, Date):
Patent: WO 9200047 A1 19920109
Application: WO 91US4466 19910620 (PCT/WO US9104466)
Priority Application: US 90542326 19900622
Designated States: AT AU BE CA CH DE DK ES FR GB GR IT JP KR LU NL SE
Publication Language: English
Fulltext Word Count: 16190

English Abstract
A process for selecting the types of cells that will grow on a structure, such as an implantable device or a cell growth surface. The implantable device may have a titanium surface. The process includes attaching a molecular monolayer to the surface of the structure. The monolayer has a functional group at its distal end. The possible function groups include CH3, CH=CH2, Br, CN, COOH, and CHOHCH2OH. The monolayer is coated with an adhesion-mediating molecule such as fibronectin. Cells then contact the coating. The character of the functional group affects the growth characteristics of the adhering or contacting cell, independently of the nature of the underlying structure. Also disclosed is a method of preparing a metallic surface such as titanium to receive a molecular monolayer. The surface is placed in hot water (40-50 °C) for 4 hours with sonication, or in boiling water for 8 hours without sonication.

Japanese Abstract
Procede de selection de types de cellules se developpant sur une surface, tel qu'un dispositif implantable ou une surface de croissance cellulaire. Le dispositif implantable peut avoir une surface en titane. Le procede consiste a fixer une monocouche moleculaire a la surface de la structure. La monocouche comporte un groupe fonctionnel au niveau de son extremite distale. Les groupes fonctionnels possibles sont CH3, CH=CH2, Br, CN, COOH, et CHOHCH2OH. On enduit la monocouche d'une molecule enduisant une adherence telle que la fibronectine. Les cellules viennent alors au contact du revetement. Le caractere du groupe fonctionnel affecte les caracteristiques de croissance de la cellule d'adherence ou de contact, independamment de la nature de la structure sous-jacente. L'invention concerne egalement un procede de preparation d'une surface metallique telle que du titane en vue de l'application d'une monocouche moleculaire. On place la surface dans de l'eau chaude (40 a 50 °C) pendant quatre heures avec sonication, ou dans de l'eau bouillante pendant 8 heures sans sonication.

29/3,AB/39 (Item 1 from file: 654)
DIALOG(R)File 654:US PAT.FULL.

02581827
Utility
USE OF ISOLATED DOMAINS OF TYPE IV **COLLAGEN** TO MODIFY CELL AND TISSUE INTERACTIONS
[Antigrowth agents]

PATENT NO.: 5,567,609
ISSUED: October 22, 1996 (19961022)

INVENTOR(s): Sarras, Jr. Michael P., Kansas City, KS (Kansas), US (United States of America)
Hudson, Billy G., Lenexa, KS (Kansas), US (United States of America)
ASSIGNEE(s): University of Kansas Medical Center, (A U.S. Company or Corporation), Kansas City, KS (Kansas), US (United States of America)
[Assignee Code(s): 36361]
APPL. NO.: 8-268,969
FILED: June 30, 1994 (19940630)

STATEMENT OF RIGHTS

The U.S. Government has a paid-up license in this invention and the right in limited circumstances to require the patent owner to license others on reasonable terms as provided for by the terms of Grants No. 01-RR06500 and AM 18381 awarded by the National Institute of Health.

FULL TEXT: 560 lines

ABSTRACT

The instant invention demonstrates that the 7S and NC1 domains of type IV **collagen** disrupts cell aggregation and tissue development. Structural changes in mesoglea, inhibition of cell proliferation, and changes in cell differentiation patterns accompanies the blockage of cell aggregates which indicate that blockage may be due to alterations in mesoglea (extracellular matrix) structure with accompanying effects on cell behavior. Type IV **collagen** has a critical role in the initial formation of mesoglea and that perturbation of mesoglea formation affects cell division, cell differentiation, and morphogenesis.
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36/3,AB/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10774773 BIOSIS NO.: 199799395918
Cloning of human homolog of cyr61 **and characterization of its biological activities.**
AUTHOR: Kolesnikova T V; **Lau L F**
AUTHOR ADDRESS: Dep. Genet., Univ. Illinois, Chicago, IL 60607**USA
JOURNAL: Molecular Biology of the Cell 7 (SUPPL.):p415A 1996
CONFERENCE/MEETING: Annual Meeting of the 6th International Congress on Cell Biology and the 36th American Society for Cell Biology San Francisco, California, USA December 7-11, 1996
ISSN: 1059-1524
RECORD TYPE: Citation
LANGUAGE: English
1996

36/3,AB/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10335978 BIOSIS NO.: 199698790896
Cyr61, **a product of a growth factor-inducible immediate early gene, promotes cell proliferation, migration, and adhesion.**
AUTHOR: Kireeva Maria L; Mo Fan-E; Yang George P; **Lau Lester F** (a
AUTHOR ADDRESS: (a)Dep. Genetics, M/C 669, Univ. Illinois Coll. Med., 900 South Ashland Ave., Chicago, IL 60607-717**USA
JOURNAL: Molecular and Cellular Biology 16 (4):p1326-1334 1996
ISSN: 0270-7306
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **cyr61** was first identified as a growth factor-inducible immediate-early gene in mouse fibroblasts. The encoded **Cyr61** protein is a secreted, cysteine-rich heparin-binding protein that associates with the cell surface and the extracellular matrix, and in these aspects it resembles the Wnt-1 protein and a number of known growth factors. During embryogenesis, **cyr61** is expressed most notably in mesenchymal cells that are differentiating into chondrocytes and in the vessel walls of the developing circulatory system. **cyr61** is a member of an emerging gene family that encodes growth regulators, including the connective tissue growth factor and an avian proto-oncoprotein, Nov. **cyr61** also shares sequence similarities with two Drosophila genes, twisted gastrulation and short gastrulation, which interact with decapentaplegic to regulate dorsal-ventral patterning. In this report we describe the purification of the **Cyr61** protein in a biologically active form, and we show that purified **Cyr61** has the following activities: (i) it promotes the attachment and spreading of endothelial cells in a manner similar to that of fibronectin; (ii) it enhances the effects of basic fibroblast growth factor and platelet-derived growth factor on the rate of DNA synthesis of fibroblasts and vascular endothelial cells, although it has no detectable mitogenic activity by itself; and (iii) it acts as a chemotactic factor for fibroblasts. Taken together, these activities indicate that **Cyr61** is likely to function as an extracellular matrix signaling molecule rather than as a classical growth factor and may regulate processes of cell proliferation, migration, adhesion, and differentiation during development.

1996

36/3,AB/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

08711737 BIOSIS NO.: 199395001088
Expression of the growth factor-inducible immediate early gene cyr61 **correlates with chondrogenesis during mouse embryonic development.**
AUTHOR: O'Brien Timothy P; **Lau Lester F** (a
AUTHOR ADDRESS: (a)Dep. Genetics, Univ. Ill. Coll. Med., 808 South Wood St., Chicago, Ill. 60612
JOURNAL: Cell Growth & Differentiation 3 (9):p645-654 1992
ISSN: 1044-9523
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Cyr61** is a growth factor-inducible immediate early gene initially identified in serum-stimulated mouse fibroblasts. It encodes a member of an emerging family of cysteine-rich secreted proteins that includes a connective tissue growth factor. We show here that **cyr61** is expressed in the developing mouse embryo and extraembryonic tissues. In the placenta, **cyr61** is expressed in regions of trophoblastic origin, including the ectoplacental cone and the trophoblastic giant cells. In the midgestation embryo, **cyr61** is expressed in the smooth muscle vessel walls of the arterial circulatory system. Most notably, expression is found in developing cartilaginous elements, including the limbs, ribs, and prevertebrae. In addition, regions of the chondrocranium and craniofacial elements, such as Meckel's cartilage, also express **cyr61** . Thus, **cyr61** transcripts is found in mesenchymal cells of both mesodermal and ectodermal origin during their differentiation into chondrocytes. The temporal and spatial regulation of **cyr61** expression and the biochemical features of its encoded protein suggest that **cyr61** may be important for the normal growth, differentiation, or morphogenesis of the cartilaginous skeleton of the embryo.

1992

36/3,AB/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

08280938 BIOSIS NO.: 000043047011
THE IMMEDIATE EARLY GENE CYR61 **ENCODES A HEPARIN BINDING PROTEIN WHOSE IN-VIVO EXPRESSION CORRELATES WITH CHONDROGENESIS**
AUTHOR: YANG G P; O'BRIEN T P; ABLER A S; **LAU L F**
AUTHOR ADDRESS: DEP. GENET., UNIV. ILL. COLL. MED., CHICAGO, ILL. 60612, USA.
JOURNAL: KEYSTONE SYMPOSIUM ON GROWTH AND DIFFERENTIATION FACTORS IN VERTEBRATE DEVELOPMENT, KEYSTONE, COLORADO, USA, APRIL 3-10, 1992. J CELL BIOCHEM SUPPL 0 (16 PART F). 1992. 104. 1992
CODEN: JCBSD
DOCUMENT TYPE: Meeting
RECORD TYPE: Citation
LANGUAGE: ENGLISH
1992

36/3,AB/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

07750351 BIOSIS NO.: 000092064072
CYR61 **PRODUCT OF A GROWTH FACTOR-INDUCIBLE IMMEDIATE EARLY GENE IS ASSOCIATED WITH THE EXTRACELLULAR MATRIX AND THE CELL SURFACE**
AUTHOR: YANG G P; **LAU L F**
AUTHOR ADDRESS: DEP. GENETICS, UNIVERSITY ILLINOIS COLLEGE MEDICINE, 808 SOUTH WOOD STREET, CHICAGO, ILLINOIS 60612.
JOURNAL: CELL GROWTH DIFFER 2 (7). 1991. 351-358. 1991
CODEN: CGDIE
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: **Cyr61** is a specific target for activation by platelet-derived growth factor and fibroblast growth factor and is inducible by the oncogene v-src. It is a member of the class of immediate early genes that includes those encoding protooncogene products, transcription factors, and cytokines. We have previously characterized the synthesis and degradation of the **cyr61** -encoded mRNA and protein. Although the deduced **Cyr61** protein sequence contains an NH2-terminal secretory signal, it is not detectable in the conditioned medium of serum-stimulated cells. We show here that in rapidly growing cell cultures, newly synthesized **Cyr61** is secreted and is associated with both the extracellular matrix and the cell surface. In contrast, **Cyr61** secreted in serum-stimulated quiescent cells is directed to the cell surface and is not incorporated into the extracellular matrix. Once associated with the extracellular matrix, **Cyr61** has a half-line of greater than 24 h, whereas intracellular and cell surface-associated **Cyr61** has an apparent half-life of approximately 30 min. Furthermore, **Cyr61** appears to bind heparin with high affinity. These observations suggest similarities among **Cyr61** , the fibroblast growth factors (heparin-binding growth factors), and the protooncogene product Int-1 and are consistent with the hypothesis that **Cyr61** plays a role in cell-cell communication involving the interaction of neighboring cells.

1991

36/3,AB/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

07750275 BIOSIS NO.: 000092063996
PROMOTER FUNCTION AND STRUCTURE OF THE GROWTH FACTOR-INDUCIBLE IMMEDIATE EARLY GENE CYR61
AUTHOR: LATINKIC B V; O'BRIEN T P; **LAU L F**
AUTHOR ADDRESS: DEP. GENETICS, UNIV. ILL. COLL. MED., 808 SOUTH WOOD ST., CHICAGO, ILL. 60612.
JOURNAL: NUCLEIC ACIDS RES 19 (12). 1991. 3261-3268. 1991
FULL JOURNAL NAME: Nucleic Acids Research
CODEN: NARHA
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: **cyr61** is an immediate early gene that is transcriptionally activated in 3T3 fibroblasts by serum, platelet-derived growth factor, fibroblast growth factor, and the tumour promoter TPA with kinetics similar to the induction of c-fos. **cyr61** encodes a secreted protein that is associated with the cell surface and the extracellular matrix,

and may play a role in cell-cell communication. We report here the complete nucleotide sequence of the mouse **cyr61** gene, which contains four short introns. The transcription start site was mapped by S1 nuclease and primer extension analyses. A 2 kb 5' flanking DNA fragment functions as a serum-inducible promoter. This DNA fragment contains a poly(CA) sequence that can adopt the Z DNA form. In addition, it contains a sequence that resembles the serum response element (SRE) originally idnetified in the c-fos promoter. We show that deletion of the **cyr61** SRE-like sequence abrogates serum inducibility. Furthermore, this SRE-like sequence is sufficient to confer serum and growth factor inducibility when linked to a basal promoter, and binds the 67 kD serum response factor in vitro. We conclude that the **cyr61** SRE functions as a serum response element and may account for the coordinate activation of **cyr61** and c-fos.

1991

36/3,AB/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

07270660 BIOSIS NO.: 000090050539
EXPRESSION OF CYR61 **A GROWTH FACTOR-INDUCIBLE IMMEDIATE-EARLY GENE**
AUTHOR: O'BRIEN T P; YANG G P; SANDERS L; **LAU L F**
AUTHOR ADDRESS: DEP. GENET., UNIV. ILL. COLL. MED., 808 S. WOOD ST., CHICAGO, ILL. 60612, USA.
JOURNAL: MOL CELL BIOL 10 (7). 1990. 3569-3577. 1990
FULL JOURNAL NAME: Molecular and Cellular Biology
CODEN: MCEBD
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: A set of immediate-early genes that are rapidly activated by serum or purified platelet-derived growth factor in mouse 3T3 fibroblasts has been previously identified. Among these genes, several are related to known or putative transcription factors and growth factors, supporting the notion that some of these genes encode regulatory molecules important to cell growth. We show here that a member of this set of genes, **cyr61** (originally identified by its cDNA 3CH61), encodes a 379-amino-acid polypeptide rich in cysteine residues. **cyr61** can be induced through protein kinase C-dependent and -independent pathways. Unlike many immediate-early genes that are transiently expressed, the **cyr61** mRNA is accumulated from the G0/G1 transition through mid-G1. This expression pattern is due to persistent transcription, while the mRNA is rapidly turned over during the G0/G1 transition and in mid-G1 at the same rate. In logarithmically growing cells, the **cyr61** mRNA level is constant throughout the cell cycle. **Cyr61** contains an N-terminal secretory signal sequence; however, it is not detected in the culture medium by immunoprecipitation. **Cyr61** is synthesized maximally at 1 to 2 h after serum stimulation and has a short half-life within the cell.

1990
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7/27 Brumback 10-12
09/495,448

CORRELATES WITH CHONDROGENESIS
AUTHOR: YANG G P; O'BRIEN T P; ABLER A S; LAU L F
AUTHOR ADDRESS: DEP. GENET., UNIV. ILL. COLL. MED., CHICAGO, ILL. 60612, USA.
JOURNAL: KEYSTONE SYMPOSIUM ON GROWTH AND DIFFERENTIATION FACTORS INVERTEBRATE DEVELOPMENT, KEYSTONE, COLORADO, USA, APRIL 3-10, 1992. J CELLBIOCHEM SUPPL 0 (16 PART F). 1992. 104. 1992 CODEN: JCBSD

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Regulation of expression of growth factor-inducible immediate-early genesCYR61 and PIP92
AUTHOR(S): Latinkic, Branko Vasilije
LOCATION: Univ. Illinois, Chicago, IL, USA
DATE: 1994 PAGES: 186 pp. CODEN: DABBBA LANGUAGE: English CITATION:Diss. Abstr. Int. B 1994, 55(5), 1749 AVAIL: Univ. Microfilms Int., Order
No. DA9426517

120185699 CA: 120(15)185699x DISSERTATION
Biochemical and functional analysis of Cyr61, the product of a growthfactor-inducible immediate early gene
AUTHOR(S): Yang, George P.
LOCATION: Health Sci. Cent., Univ. Illinois, Chicago, IL, USA
DATE: 1993 PAGES: 129 pp. CODEN: DABBBA LANGUAGE: English CITATION:Diss. Abstr. Int. B 1994, 54(8), 3953 AVAIL: Univ. Microfilms Int., Order
No. DA9335171

120126810 CA: 120(11)126810d DISSERTATION
Characterization and expression analysis of the growth factor-inducible immediate-early gene cyr61
AUTHOR(S): O'Brien, Timothy Paul
LOCATION: Health Sci. Cent., Univ. Illinois, Chicago, IL, USA
DATE: 1992 PAGES: 126 pp. CODEN: DABBBA LANGUAGE: English CITATION:Diss. Abstr. Int. B 1993, 53(8), 3911 AVAIL: Univ. Microfilms Int., Order
No. DA9238021

SN 09/495,448

Extracellular matrix signaling protein CYR61 is downregulated in humanprostate and kidney cancer
Froschermaier, S.E.; Schmidt, U.; Eissrich, C.; Stade, J.; Pilarsky, C.P.; Haase, M.; Faller, G.; Kirchner, T.W.; Wirth, M.P.
European Association of Urology XIVth Congress 9810518 Stockholm(Sweden) 21-25 Mar 1998 European Association of Urology
EAU Congress Office, P.O. Box 204, 6600 AE WIJCHEN, The Netherlands;
phone: +31 (0)24 6452510; fax: +31 (0)24 64550769; URL: http://www.bpc.nl/uroweb/, Abstracts available.
Poster Paper No. 527

Title: N-SYNDECAN - STRUCTURE AND FUNCTION OF A TRANSMEMBRANE HEPARAN- SULFATE PROTEOGLYCAN (Abstract Available)
Author(s): CAREY DJ
Corporate Source: WEIS CTR RES,GEISINGER CLIN/DANVILLE//PA/17822
Journal: PERSPECTIVES ON DEVELOPMENTAL NEUROBIOLOGY, 1996, V3, N4, P331-346 ISSN: 1064-0517

Title: EFFECTS OF FIBRIN ON THE ANGIOGENESIS IN-VITRO OF BOVINE ENDOTHELIAL-CELLS IN COLLAGEN GEL (Abstract Available)
Author(s): TAKEI A; TASHIRO Y; NAKASHIMA Y; SUEISHI K
Corporate Source: KYUSHU UNIV 60,FAC MED,DEPT PATHOL 1,HIGASHI KU,3-1-1 MAIDASHI/FUKUOKA 812//JAPAN/
Journal: IN VITRO CELLULAR & DEVELOPMENTAL BIOLOGY-ANIMAL, 1995, V31, N6 (JUN), P467-472 ISSN: 1071-2690